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Gastrointestinal complications after cardiac surgery

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SUMMARY

Gastrointestinal complications after cardiac surgery are relatively rare entities but carry a high mortality. We identified over 70 articles written since 2010 using the PubMed database. We included 40 in our review. The most common complications include paralytic ileus, gastrointestinal bleeding, and bowel ischemia. Patients who undergo cardiac procedures are at risk for poor perfusion of the gastrointestinal tract and, thus, at risk for resulting complications. Risk factors for these complications include peri-operative use of vasopressors, prolonged operative time, and the time of cardiopulmonary bypass. Presentation of gastrointestinal complications tends to differ as patients after open heart surgery can remain intubated, and exams can be limited. Early recognition and aggressive therapy are paramount. We aim to provide a review that will help the reader get familiar with the most common gastrointestinal complications that can negatively affect outcomes after cardiac surgery.

INTRODUCTION

Gastrointestinal (GI) complications after cardiac surgery are relatively rare entities but carry high mortality.[1] This article aims to provide an update on a prior comprehensive review of this topic and its development since 2010.

METHODS

The PubMed database was searched using the following keywords: *cardiac surgery, CABG, gastrointestinal complications, acute cholecystitis, bowel ischemia, mesenteric ischemia, diverticulitis, peptic ulcer, liver failure, and GI bleeding*.

The search yielded 70 articles. 59 were evaluated in detail, and 40 were included in our review. The excluded articles included animal studies, older review articles, and papers irrelevant to post-operative GI complications in cardiac surgery.

GASTROINTESTINAL COMPLICATIONS

Since 2010, over three dozen papers have been written on GI complications after cardiac surgery (table 1). Seven major retrospective studies assessed the frequency and risk factors of GI complications following a cardiac operation.[2–8] The most commonly cited complications were paralytic ileus, upper GI bleeding, mesenteric ischemia, acute cholecystitis, and acute pancreatitis. Mortality of GI complications across the studies above ranged from 10% to 38%.[2–8] One study focused on emergency general surgery consultation rates as opposed to actual rates of the pathology.[7]

Major pre-operative risk factors were a history of hypertension (HTN),[6] renal insufficiency,[3 6 8] advanced age,[5 8] congestive heart failure,[5 6] and a history of prior GI pathology.[4 5]

The peri-operative risk factors included peri-operative use of vasopressors,[4 6 8] prolonged skin-to-skin time,[3] cardiopulmonary bypass (CPB) time,[3 5 8] and cross-clamp time.[5]

Patients undergoing complex or combined cardiac procedures such as valve surgery and coronary artery bypass graft (CABG) were also at increased risk.[5–8] Post-operative risk factors included prolonged mechanical ventilation,[4 5] and the need for renal replacement therapy.[4 8]

The complication with the highest mortality was mesenteric ischemia, approaching up to 100%.[2]

ACUTE CHOLECYSTITIS

Since 2010, several studies have discussed rates of acute cholecystitis following cardiac surgery, with an overall rate of 0.21%–0.53%.[3 5–8] Acute cholecystitis forms up to 23% of GI complications following cardiac procedures.[6] None of the studies cited in this section focused solely on acute cholecystitis. In a study by Dong *et al*, acalculous cholecystitis was slightly more prevalent (60%) than calculous, while in a review by Ashfaq *et al*, it was at least 67%. In a study by Gulkarov *et al*, all patients suffered acalculous cholecystitis, and interestingly, all had accompanied GI bleeding at some point following cardiac surgery.[6]

In the same series, two out of three patients presented with right upper quadrant (RUQ) abdominal pain, while the third patient was diagnosed after developing jaundice and rising liver function test.[6] A study by Marsoner *et al* reports typical presentation in most patients with fever and RUQ abdominal pain.[3] Disease-specific mortality ranged from 0% to 33%.[3 6 8] Early recognition and prompt management are paramount. The most common findings were ultrasound findings of gallbladder wall thickening with or without surrounding pericholecystic fluid.[6 7]

Management of acute cholecystitis includes broad-spectrum antibiotics and prompt source control such as percutaneous cholecystostomy or cholecystectomy. Surgical treatment may be preferential to the percutaneous approach, as with the latter, some patients may fail to improve.[6] Even though laparoscopic cholecystectomy is the gold standard, most patients who suffer from cholecystitis after cardiac surgery undergo open cholecystectomy.[3 8] This is likely due to the inability to tolerate pneumopritoneum soon after cardiac surgery as it negatively affects cardiovascular hemodynamics. In a study by Marsoner *et al*, 12 out of 18 patients underwent open cholecystectomy, while 2 were managed laparoscopically. Three patients received

Table 1 Gastrointestinal complications after cardiac surgery*

Study	Year	Patients	GI complications (absolute number)	Mortality†	Pancreatitis	Acute cholecystitis	Diverticulitis	Ileus/SBO	Bowel ischemia	UGIB	Liver failure	LGIB	PPU
Ashfaq *et al*[7]	2007–2010	914	2.5% (23)	10%		13% (0.3%)		17.4% (0.43%)	8.7% (0.21%)				
Chung *et al*[10]	2005–2010	986		15.5%	5.9%								
van Diepen *et al*[11]	2017–2018	1625		4.3%–4.8%						3.9%–4.8%			
Krawiec *et al*[14]	2002–2012	9017		8.8%						1.5% (138) 1.01% included in study (91)			
Fan *et al*[16]	1998–2005	6316		47.6%						0.3%			
Marsoner *et al*[3]	2008–2013	4883	2.9% (142)	12.1%	28.8% (0.84%)	12.6% (0.37%)		9.9%/3.52% (0.29%/0.10%)	11.2% (0.32%)	5.6% (0.16%)		7% (0.21%)	
Vohra *et al*[4]	2001–2005	2320	2.8% (65)	21.5%				23%/7.6% (0.64%/0.21%)	18.4% (0.51%)	24.6% (0.69%)		12.3% (0.34%)	
Viana *et al*[8]	2001–2011	5382	1.1% (61)	33%		1.6% (0.02%)			18% (0.2%)	21% (0.23%)	12% (0.13%)	8.1% (0.09%)	8.1% (0.09%)
Dong *et al*[5]	2004–2010	2349	1.4% (33)	15.2%		15.2% (0.21%)		33.3% (0.46)	6.1% (0.09%)	27.4% (0.38%)			6.1% (0.09%)
Gulkarov *et al*[6]	2003–2005	565	2.3% (16)	38%		23% (0.53%)	7.7% (0.18%)		23% (0.53)	69.2% (1.6)			
Sever *et al*[2]	2009–2012	1360	0.21% (29)	27.5%				41.4% (0.08%)	27.5% (0.06%)	24.1% (0.05%)		6.9% (0.02%)	
Nilsson *et al*[32]	1996–2011	18 879		59%					0.09%				
Sakamoto *et al*[31]	2010–2017	211 900		77% (NOMI)					0.26%, 0.06% NOMI				
Ariyaratnam *et al*[30]	1999–2014	13 853		60%					0.62%				
Warwick *et al*[29]	1997–2012	18 325		87%					0.49%				
Lim *et al*[24]	2007–2015	9445		57.5%					0.4%				
Pang *et al*[27]	1999–2010	9925		71%					0.31%				
Takeyoshi *et al*[26] ‡	2014–2020	100		100%					0.8%				
Eris *et al*[25]	2005–2013	6037		67%					0.86%				
Mothes *et al*[24]	2005–2012	9385		68%					1.15%				
Klingele *et al*[21] / Groesdonk *et al*[22]	2010–2011	865		–					9.0%				
Krasivskyi *et al*[20]	2011–2015	7525	2.33% (175)	89.7% (bowel ischemia only)					22.2% (0.6%)				
Guillaume *et al*[19]	2007–2013	4948		64% and 83% (28 vs 90 days)					0.66				
Sastry *et al*[17]	2006–2011	10 409		100%					0.3%				
Soylu *et al*[18]	2010–2016	7606		61.2%					0.4%				

A total of 364 920 patients.
*In windows with two percentage values, the first represents proportion to other GI complications in the series and the second, placed in brackets, rates in the overall study population.
†Either overall from GI complications or complications specific in focused studies.
‡Hemodialysis patients only.
GI, gastrointestinal; LGIB, lower GI bleeding; NOMI, non-occlusive mesenteric ischemia; PPU, perforated peptic ulcer; SBO, small bowel obstruction; UGIB, upper GI bleeding .

Table 2 Pre-operative risk factors for development of bowel ischemia after cardiac surgery[23–25 29–32 35]

Univariate	Multivariate
Age	IABP
Cardiogenic shock	Age
Renal insufficiency	PVD
Pressors	Pressors
IABP	Lactic acid >5 mmol/L
Steroids	
Emergency surgery	
NYHA IV	
Liver cirrhosis	

IABP, intra-aortic balloon pump; NYHA, New York Heart Association; PVD, peripheral vascular disease.

a cholecystostomy tube; in most of them, it was a bridge to a definitive operation.[3]

ACUTE DIVERTICULITIS

We found one study and one case report regarding acute diverticulitis following cardiac surgery.[6 9] The reason for this scarce amount of literature is likely the overall rarity of this condition, as both papers only produced two patients with this diagnosis. The incidence was 0.18% in patients undergoing mitral valve surgery.[6] A case report discussed a single patient who underwent a left ventricular aneurysm repair on CBP.[9] Both patients survived. The patients were diagnosed with acute diverticulitis after developing abdominal pain between post-operative days 5 and 8.[6 9] Both were managed non-operatively with intravenous antibiotics alone,[6] or intravenous antibiotics and percutaneous drainage,[9] and discharged home.

ACUTE PANCREATITIS

Only two studies have investigated the rates of acute pancreatitis since 2010. We found only one article focusing solely on rates and risk factors of acute pancreatitis.[10] This study by Chung *et al* focused strictly on patients after valve replacement and yielded relatively high rates of 5.9%,[10] compared with a study by Marsoner *et al*, with an overall rate of 0.84%.[3] The significant difference could be explained by lower serum marker threshold for diagnosis in the paper by Chung *et al*, as neither of the studies required patients to have abdominal pain for diagnosis.[3 10] Patients who suffered acute pancreatitis after cardiac surgery had a mortality of 0%–15.5%,[3 10] and an extended hospital stay.[10]

Table 3 Peri-operative and post-operative risk factors for development of bowel ischemia after cardiac surgery[19 24 25 29–32 34 35]

Univariate	Multivariate
Pressors use	Cr >2.3 mg/dL
LA >3	IABP
CK-MB	MV >24 hours
Prolonged MV	CVA
Blood products	Arterial fibrillation
Re-operation	CPB
CPB time	

CK-MB, creatine-kinase isoenzyme MB ; CPB, cardiopulmonary bypass; Cr, creatinine; CVA, cerebrovascular accident; IABP, intra-aortic balloon pump; LA, lactic acid; MV, mechanical ventilation.

The average time from index cardiac operation to diagnosis was 9 days.[3] The criteria for diagnosis of acute pancreatitis varied and included lipase of >180 U/L, lipase of more than three times the patient's baseline or >60 U/L if the patient had concurrent abdominal pain,[10] or leukocytosis and elevated lipase >300 U/L regardless of the presence of abdominal pain.[3] Independent risk factors, identified through multivariate logistic regression, included pre-operative HTN, chronic kidney disease, and intra-operative use of norepinephrine.[10] The most common symptoms were abdominal pain followed by nausea and vomiting. All patients were managed non-operatively with bowel rest and the addition of oral pancreas enzyme. Somatostatin was added in severe cases.[10]

PEPTIC ULCER

A search for articles regarding peptic ulcer disease yielded three relevant papers.[5 8 11] As there is an overlap with upper GI bleeding (UGIB), more studies will be discussed in the following section.

The perforated peptic ulcer rate was 0.09%, and no mortalities were observed. It constituted up to 6.1% of all GI complications, nearly identical to our prior review.[1 5] In a study by Viana *et al*, only the total number of patients with perforated viscus was listed (8.1% of all patients with GI complications), and the majority were due to perforated duodenal ulcers,[8] and carried 60% mortality. In the series by Dong *et al*, nearly half of the patients with UGIB had pre-existing gastroduodenal ulcers.[5]

An ad hoc analysis was performed on 823 Canadian patients who underwent cardiac surgery and were previously randomized into a PEPTIC trial (efficacy of the proton pump inhibitors (PPIs) vs histamine-2 receptor blockers (H2B) for ulcer prophylaxis trial). The PEPTIC trial evaluated the efficacy of PPIs and H2B among patients in various intensive care units. There was no statistically significant difference in clinically relevant GI bleeding in cardiac surgery patients between the ones receiving PPIs versus H2B.[11] Additionally, there was no difference in 90 days mortality.[11]

A perforated peptic ulcer is most commonly managed with open Graham's patch repair as other, more extensive procedures, such as distal gastrectomy with Billroth reconstruction or vagotomy with drainage procedure, have fallen out of favor due to significant morbidity. However, these procedures still have a role in cases where Graham's patch is not feasible. Graham's patch repair consists of patching the perforation with a tongue of omentum. In a modification of the technique, the defect is first closed primarily prior to placement of the omental patch. In order to reduce the morbidity of laparotomy, this procedure can be performed laparoscopically.

UPPER GI BLEEDING

Since 2010, several studies have focused on GI bleeding following cardiac surgery.[12–15] Additionally, GI bleeding rates as a part of GI complications were investigated in a few papers.[2–6 8] Postcardiac surgery GI bleeding rates varied from 0.07% to 1.6%, affecting up to 69.2% of patients who suffer post-operative GI complications.[2–6 8 11 12 14] Upper GI bleeding carries high mortality, up to 47.6%.[16]

Risk factors for UGIB included advanced age,[14 16] prolonged mechanical ventilation,[12] elevated international normalized ratio (INR),[14] and CPB duration.[16] Patients who underwent isolated CABG were less likely to suffer UGIB.[14]

PPIs were routinely used in several series,[3 16] as they were clearly shown to have a protective effect.[16] Additionally, a study by Fan *et al* compared the effectiveness of an early transition to

Box 1 Prediction model for development of bowel ischemia after cardiac surgery[23]

d=3.85×pre-operative intra-aortic balloon pump support+1.91×re-exploration for bleeding+1.86×post-operative need of >1 packed red blood cells+1.52×post-operative serum lactate >5 mmol/L+1.07×post-operative levosimendan (ionotropic agent) therapy+0.77×post-operative need for norepinephrine >0.1 µg/kg/min+0.36×post-operative loss of sinus rhythm–0.32 (constant of the equation)

For each item 0=no, 1=yes

Discrimination value=1.18

oral PPIs. Oral PPIs are more cost-effective and have the same efficacy as the intravenous form. The study showed no difference in rates of post-operative bleeding or the development of asymptomatic ulcers evaluated by routine post-operative endoscopy.[16]

In patients who also underwent pre-operative esophagogastroduodenoscopy (EGD), if found to have an ulcer, elective cardiac surgery was postponed until healing was verified.[3]

UGIB was much more common than lower GI bleeding, and in certain series accounting for 100% of all post-operative GI hemorrhagic complications.[6] The most common source was a duodenal ulcer, followed by a gastric ulcer.[3 12 14] Patients most commonly developed melena, followed by coffee ground emesis and hematemesis.[12]

The majority of patients were managed non-operatively or with endoscopic intervention.[2 3 5 6 14] Management of GI bleeding includes blood transfusion and urgent endoscopic evaluation. If a treatable lesion is identified, interventions, such as epinephrine injection or clip application, can be used. Patients who rebleed after initial stabilization should undergo repeat EGD. Additionally, CT or conventional angiography can be used to localize and embolize the culprit vessel, most commonly the gastroduodenal artery (GDA). Emergency surgery for control of upper GI hemorrhage is reserved for cases that fail therapies discussed above or become unstable and do not respond to resuscitation. Surgical intervention includes oversawing the ulcer with or without ligation of the GDA.

Lower GI bleeding

A search for papers regarding lower GI bleeding following cardiac surgery yielded three results. All studies evaluated GI complications after cardiac surgery in general.[2–4] Lower GI bleed incidence ranged between 0.02% and 0.34%, forming up to 12.3% of GI complications.[2–4] Mortality was 10% in one series.[3]

The most common source of lower GI bleeding were hemorrhoids, followed by diverticular bleeding.[3] Most patients were managed conservatively and did not require emergent surgery.[2 3]

Table 4 General recommendations for peri-operative care after cardiac surgery

Optimize pre-operative risk factors*	Limit use of vasopressors
Limit duration of CPB and cross-clamp time as technically feasible	Maintain normothermia
Judicious volume resuscitation	Routine use of H2B or PPI

*See each chapter for relevant pre-operative risk factors.
CPB, cardiopulmonary bypass; H2B, histamine-2 receptor blockers; PPI, proton pump inhibitor.

BOWEL ISCHEMIA

Over the past decade, most of the attention in terms of post-cardiac surgery GI complications was focused on bowel ischemia.[17–32] The incidence of mesenteric ischemia among cardiac surgery patients ranged from 0.06% to 1.15%. Bowel ischemia is a condition with very high mortality of 57.5%–100%. Emboli are the most common etiology of acute bowel ischemia in the general population. However, hypoperfusion leading to non-occlusive mesenteric ischemia is far more common in post-cardiac surgery patients, making it the leading etiology in this subgroup.[21 23 26 31 33 34] The focus in the majority of studies is on risk factors for mesenteric ischemia and its early recognition.

Pre-operative risk factors for small bowel ischemia (table 2) include peripheral vascular disease (PVD),[25 29 30 32 35] advanced age,[25 29–31 34 35] cardiogenic shock,[25 32 35] blood product use,[23 29] pre-operative vasopressors requirements,[23 25 31] renal failure,[24 25 31] and pre-operative need for intra-aortic balloon pump (IABP).[23 25 30 35]

The most recent study that compared rates of bowel ischemia in patients who underwent CABG on versus off CPB found significantly higher rates in the on-pump group.[18] Moreover, the on-pump group also had much higher mortality, with survival of only 7%.[18]

Among other peri-operative and post-operative risk factors (table 3), the most significant were prolonged ventilatory support >24 hours,[25 32 35] need for post-operative vasopressors,[25 34 35] blood product use,[19 29 35] IABP,[25 35] and persistently elevated lactate.[25 34] Additional risk factors included elevated creatinine >2.3 mg/dL,[32 35] and reoperation.[29 35]

A few papers investigated serum markers as a potential tool for early recognition of bowel ischemia.[16 21 22 36 37] Lorusso *et al* analyzed several articles to evaluate which serum markers correlate with bowel ischemia. They found that D-lactate of 0.033 g/L had an 81% positive predictive value. Intestinal fatty acid binding protein (iFABP) also strongly correlates with this diagnosis.[36] However, iFABP was absent with limited small bowel involvement.[36] Glutathione S-transferase alpha levels strongly detected the presence of ischemic bowel and, in combination with transaminases, could differentiate between occlusive and non-occlusive etiology.[36]

Similar biomarkers were evaluated by Hong *et al*: smooth muscle actin (SMA), D-lactate, and iFABP in a prospective trial evaluating levels of these substances prior to undergoing laparotomy for suspected bowel ischemia.[33] All patients with positive laparotomy had elevated SMA.[33] Rising D-lactate after initial laparotomy was associated with a fatal outcome.[33] Dohle *et al* evaluated lactate, glutathione transferase, and iFABP in patients with suspected bowel ischemia.[37] Out of 18 patients who underwent laparotomy, only 50% had ischemic bowel.[37] The only marker that differed between patients with positive and negative laparotomy was iFABP.[37]

Certain procalcitonin levels were also strongly associated with non-occlusive mesenteric ischemia.[21 36] While the study by Klingele *et al* found levels >6.6 ng/mL to have sensitivity and specificity over 90%,[21] Lorusso *et al* reported levels as low as 0.57 ng/mL to be associated with intractable gut ischemia.[36] Lactic acid >2 mmol/L associated with prolonged opioid consumption was also an independent predictor of small bowel ischemia.[20]

In addition to evaluating biomarkers, several studies proposed a scoring system to aid with the timely recognition of bowel ischemia.[22 30 38] While the majority focused on clinical factors, Zogheib *et al* created a scoring system that comprised laboratory values—levels of aspartate aminotransferase >449 IU/L,

lactate >4 mmol/L, procalcitonin >4.7 µg/L, and myoglobin >1882 µg/L.[38] The cut-off value achieved a sensitivity of 85% and a specificity of 94%.[38] Ariyarantnam *et al* created an equation from logistic regression parameters that included age, New York Heart Association Functional Classification 4, post-operative atrial fibrillation, use of IABP, female gender, and peripheral arterial disease.[30] With these parameters, the model's sensitivity was 79%, and specificity was 61% for the detection of bowel ischemia.[30] Similarly, Groesdonk *et al* developed a prediction model (box 1). with similar sensitivity and superior specificity of 93.8% with variables that, among others, included vasopressor support, post-operative blood transfusion, and the need for re-exploration.[23]

Unfortunately, there has not been much of a positive change in terms of survival of post-operative bowel ischemia in the past decade. Given very high mortality, it is paramount to maintain a low threshold for further diagnostic evaluation when bowel ischemia is suspected. In patients who are stable or have only non-specific symptoms initial evaluation starts with CT angiography.[17 19–21 23 25] Conventional angiography is considered gold standard in certain countries.[17 21 23 26] The main benefit of conventional angiography is that it can be both diagnostic and therapeutic.[17 21 25 26] Early operative exploration should be considered in patients with high clinical suspicion as patients without intervention have higher mortality.[27 30] Pang *et al* even argued that angiography and CTA should be forgone as it can delay definitive surgical intervention.[27] In the current era, laparoscopic exploration might be the best diagnostic tool as it is feasible,[39] readily available, and has lower morbidity than traditional laparotomy. Additionally, it can be performed at the bedside in the intensive care unit setting if needed.

Liver failure

Following cardiac surgery, a minor hepatic dysfunction, such as mild transaminitis, is common, affecting up to 40% of patients.[40] It is generally self-limiting.[40] Liver failure, however, carries high mortality, up to 50%.[5] When combined with multiorgan failure, mortality reaches 90%.[41] Liver failure comprises up to 12% of GI complications.[5 8]

Most patients who develop acute liver failure do so around post-operative day 10.[41] Acute liver failure is marked by elevated bilirubin, INR, and liver enzymes. Patients' symptoms can range in severity, including jaundice, malaise, RUQ pain, ascites, and encephalopathy.

Treatment of acute liver failure is mainly supportive and comprises respiratory support if needed, judicious volume resuscitation, correction of coagulopathy, and optimal nutrition. A study by Komardina *et al* evaluated the use of a plasma filtration device, specifically in patients with liver failure following cardiac surgery.[41] This device is effective in removing unconjugated bilirubin and bile salts from plasma.[41] Although there was no comparison group, plasma filtration appeared to have a positive effect on hemodynamics, and the studied group achieved a survival of 23%.[41] Unfortunately, these modalities may not be available in most centers.

DISCUSSION

GI complications after cardiac surgery remain a rare entity that unfortunately carries high mortality. Among the most lethal ones are mesenteric ischemia, GI bleed, and liver failure. Cardiac surgery patients possess specific challenge as they often remain intubated in the early post-operative period, cannot verbalize their complains, and may not develop the usual clinical symptoms. Early diagnosis and appropriate management are mandatory and may positively affect the outcomes. Routine laboratory workup in the immediate post-operative period may allow for the detection of markers suggestive of underlying GI pathology. Further studies are encouraged to further improve understanding of GI complications that can eventually lead to better outcomes.

Table 4 depicts several recommendations that were deducted from reviewed studies and could decrease the risk of post-operative GI complications.

Limitations

Our review article has two limitations. First, due to the nature of our topic, the majority of included studies were retrospective; thus, any recommendations based on these studies have inherently lower levels of evidence. Second, there is a possibility that some studies were missed during our search despite using several pertinent keywords.

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